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Acute Myocardial Infarction

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INTRODUCTION

As a result of the tremendous advances in the prevention and treatment of many diseases, and of the increasing span of life, cardiovascular disease now greatly outnumbers all others and causes more than half the deaths after the age of fifty (7). Acute heart conditions require emergency treatment more than do those of any other organ, and by the application of well established rules and the utilization of recent advances in the field of cardiology, efficient management will save many lives and hasten recovery in others.

Although there are a number of pathophysiological factors which may produce an acute cardiac condition, acute myocardial infarction is, at present, the most prevalent and lethal, being responsible for more than 60% of medical cardiac deaths (2).

This article, therefore, shall be confined to the diagnosis and management of acute myocardial infarction and several of its more common complications.

Acute myocardial infarction is the clinical syndrome characterized by prolonged substernal oppression or pain, a fall in blood pressure and other manifestations of shock, accompanied by characteristic progressive electrocardiographic changes, fever, leucocytosis and an increased sedimentation rate.

PATHOGENESIS AND ETIOLOGY

The basic cause of myocardial infarction is an inadequate coronary blood supply, usually the product of occlusion by thrombosis, subintimal hemorrhage, or rupture of an atheromatous abscess. Embolism from left auricular mural thrombi in mitral stenosis, from the auricular appendage in auricular fibrillation or from thrombotic vegetations in bacterial endocarditis may cause occlusion and subsequent infarction. Prolonged effort or excitement superimposed on a previously compensating circulation may cause infarction without the occurrence of fresh coronary arterial lesions. Although arteriosclerosis is the most frequent etiology, arteritis due to syphilis, rheumatic fever, systemic infections or thromboangiitis obliterans may occasionally be responsible. The left anterior descending branch of the left coronary is most frequently affected, producing the "anterior" type of infarct in which the anterior wall of the left ventricle, the adjacent area of the septum and the apex are involved. Less common is involvement of the posterior descending branch of the right or left coronary, causing a "posterior" infarct, viz. the posterior portions of the I. V. septum and the diaphragmatic and posterior portions of the right and left ventricles.

The resulting myocardial infarction is the lesion responsible for the clinical syndrome observed.

INCIDENCE

Cardiovascular disease now causes more than fifty per cent of deaths after the age of fifty. In a review of 323 medical cardiac deaths by the Mayo Clinic in Rochester, the following etiological classification was produced (2):

Disease Group	% of Total Medical Cardiac Deaths
1. Coronary heart disease	63.5%
2. Hypertensive heart disease (1)	11.1
3. Valvular heart disease	16.4
4. Cor pulmonale	5.3
5. Miscellaneous	3.7

(1) A combination of coronary and hypertensive heart disease was classified as coronary heart disease. Thus, only "pure" hypertensive heart disease (i.e. absence of pathological evidence of coronary disease) is included in disease group 2.

The overwhelming predominance of coronary disease amounting to nearly two-thirds of cases is especially noticeable. A record of hypertension was present in approximately 30% of cases labelled coronary disease, this fact emphasizing the importance of hypertension as a possible etiological factor in the production of coronary disease.

Acute myocardial infarction affects men approximately six times as often as women, and occurs with increasing frequency in the 6th to 8th decades. However, as many as 2% occur before the age of 30.

DIAGNOSIS

1. Clinical Symptoms and Signs

The onset of acute myocardial infarction is denoted by the abrupt appearance of severe persistent pain at rest, during sleep, or in the course of routine activity. It may be described as a sharp, viselike, squeezing, or choking sensation, and is usually substernal or precordial but may be situated in, or radiate to, the back of the chest, the neck or the arms. Occasionally the pain initially is relatively mild, increasing in severity and becoming intense only after 15 to 30 minutes. In over half the cases, the process of thrombosis is initiated by a subintimal hemorrhage, which leads to secondary changes in the intima and eventual coronary occlusion. This process of thrombus formation may take days or weeks. During this period, the patient may experience severe episodes of angina or recurrent pain at rest, the so-called premonitory phase of an occlusion. Instead of pain, the patient may complain of severe burning sensations or feelings of tightness with nausea, vomiting and profuse perspiration. Varying degrees of shock may be present. The sudden onset of paroxysmal tachycardia or pulmonary oedema is occassionally the first evidence of an acute coronary occlusion.

Examination may reveal gallop rhythm, embryocardia, tic-toc rhythm, or pulmonary congestion. Ventricular extra-systoles, partial heart block, atrial fibrillation, atrial tachycardia or ventricular tachycardia may be present. A pericardial friction rub is heard in approximately 10% of the cases, but usually does not appear until the second or third day of the illness. Although generally persisting for only a day or two, it may remain up to a week. The blood pressure may be normal at first, or it may drop precipitously. In those with prior hypertension it may be profoundly lowered without reaching absolute low values. The pulse may not be detectable at the wrist and only barely palpable over the carotids. In most patients the ventricular rate is above 100/min: however, particularly in those who are in shock, the rate may be slow because of vagal inhibition or A-V block. The pain almost always persists for at least a half hour and usually for several hours; after it subsides a dull ache or heaviness remains. With the onset of the attack the patient frequently becomes cyanotic, pale or ashen grey, with cold moist skin, is dyspneic, and in his anguish is apprehensive and fearful of impending death.

A moderate fever generally develops within 48 hours, the rise in temperature being roughly proportional to the severity and extent of the infarction. It usually does not exceed 102°F. A higher temperature should suggest the possibility of complications such as pneumonia or other infections.

A leucocytosis of 12000-15000 cells/cu. mm. is commonly observed as well as an elevated sedimentation rate, often to 50

mm. per hour or more, the latter occurring within the first 3 or 4 days after infarction.

2. Electrocardiogram

The E.C.G. is of major importance in establishing the diagnosis of acute myocardial infarction. The characteristic QRS changes, ST segment displacements and T wave inversions are seen in almost every case although they may not appear for several days or even a week. It is important to take serial tracings every few days to establish the progressive nature of the electrocardiographic changes and thus differentiate infarctive aberrations from the non-specific ECG abnormalities of transient myocardial ischaemia, myocarditis, pericarditis and the non-cardiac conditions such as cholecystitis, perforated peptic ulcer, or pulmonary infarctions.

The ECG may show RS-T depression and T wave inversion, indicating coronary insufficiency with myocardial ischaemia. These changes, however, are replaced by a Q wave and ST segment elevation as soon as the occlusion has become complete. A Q wave usually appears in one or more leads, depending upon the site of the infarction. During the following days and weeks, serial tracings reveal a progressive return of the RS-T segment to normal and an inversion of the T wave, resulting in a Q- inverted- T pattern, which may persist for months or years.

DIFFERENTIAL DIAGNOSIS

The diagnosis of acute myocardial infarction can be made with great accuracy, if the cardinal criteria of the conditions (see definition) are kept clearly in mind, and the other conditions exhibiting similar symptomatology are briefly reveiwed.

Acute abdominal conditions may simulate cardiac pain, but usually may be differentiated by the presence of specific abdominal signs, such as localized pain, tenderness or a palpable mass in the presence of normal heart sounds and electrocardiogram. These conditions include: acute cholecystitis, appendicitis, perforated peptic ulcer, acute pancreatitis, acute mesenteric thrombosis, renal colic, intestinal obstruction, cardiospasm, and hiatus hernia.

Extracardiac thoracic conditions which must be ruled out are: acute idiopathic pericarditis, dissecting aneurysm, acute spontaneous pneumothorax, spontaneous mediastinal emphysema, herpes zoster, paroxysmal tachycardia and pulmonary embolism. It is important not to mistake pericarditis for coronary occlusion, since anticoagulant therapy is contraindicated in the former because of the danger of producing hemopericardium.

Once the cardiac origin of the pain has been established, the *type* of acute cardiac episode must be determined. The character, intensity, and duration of the pain may help to distinguish between attacks of coronary insufficiency including angina pectoris, and coronary occlusion. The prolonged, severe pain of occlusion, unrelieved by nitrites, contrasts with the briefer pain of angina which is relieved by nitroglycerine. Occasionally, however, coronary occlusion is associated with only mild pain and no other symptoms, whereas, insufficiency may cause severe pain.

The entire clinical picture and ECG changes should be considered before a definite diagnosis is made, and this frequently is possible only after observation for several days.

Any patient, therefore, who experiences sudden precordial pain, diagnosed to be cardiac in origin, should be put at rest and treated as a case of potential infarction, even though many of these cases do not progress to coronary occlusion with infarction.

MANAGEMENT

1. Relief of Pain

If it is decided that the patient has suffered a coronary thrombosis, attention must be paid first to the relief of pain. Morphine sulphate, 15 mg. subcutaneously, should be administered every 15 to 30 minutes until the desired effect is obtained, the total dose, however, not exceeding 65 mg. The arm may be elevated and massaged to increase the rate of absorption.

Little advantage is to be gained by the use of intravenous morphine, because of the rapid absorption hypodermically, and the reactive hazards involved in intravenous administration, such as acute respiratory depression. Indeed, the abrupt onset of undesirable side effect using intravenous drugs in the presence of coronary thrombosis, has made Levine suspect that these compounds act as a trigger in producing ventricular fibrillation or cardiac arrest in a susceptible heart (6).

The possibility of inducing distention, obstipation, nausea, and vomiting with morphine is great, and the drug should be discontinued with the onset of these symptoms.

Demerol, 75-100 mg., may be employed in the presence of any contra-indication to morphine, e.g. senility.

Nitroglycerine should not be used since it does not relieve the pain of occlusion, and may further lower blood pressure and reduce coronary blood flow.

2. Rest

Complete physical and mental rest is essential. The patient should be spared all needless effort through the use of good nursing care.

The vast majority of patients can be cared for at home, unless conditions there are unsatisfactory. Visitors other than the immediate family should be banned. Patients with coronary occlusion are apprehensive and discouraged; thus, it is extremely important to encourage them, stating frankly their condition, and that most patients make a satisfactory recovery and continue to lead a relatively normal life.

Up to 6 weeks of bed rest and restricted activity are indicated, depending upon the extent of the infarction, to allow a firm scar to form and thus prevent the production of ventricular aneurysm or rupture. Absolute bed rest with no muscular effort the first week, is followed the second week by leg massages 2 or 3 times daily to discourage venous thrombosis. The patient's activities in bed are begun in the 3rd or 4th week, including passive, then active leg movements and self-feeding. During the second month, increasing periods in a chair are allowed, followed by walking and other activities as tolerated. Optimum activity is usually reached in 3 to 6 months.

More recent observers, however, have emphasized the fact that prolonged bed rest is often unnecessary in acute coronary occlusion, and may have harmful effects. It has been shown that recumbency tends to increase cardiac work by raising the circulating blood volume. In addition to producing serious psychological tensions, prolonged bed rest causes generalized loss of muscular and vascular tone, constipation, and distention, and predisposes to venous thrombosis. The individual patient's progress and the physician's clinical judgement, should ensure a happy medium between these two extremes.

3. Oxygen Therapy

Oxygen is indicated in:

(1) all moderate or severe cases;

(2) always when the pain is resistant to other measures;

(3) in the presence of cyanosis.

A tent usually induces less apprehension and provides a cooler atmosphere than

does a mask, although the latter is more efficient in raising alveolar oxygen levels. Oxygen tends to relieve dyspnea, lessen pain and slow the heart rate.

4. Diet

A low caloric intake of approximately 1000 calories, largely fluids, is sufficient for the first 2 or 3 days. A urinary output of 1500 c.c. daily is desirable. Salt should be restricted until danger of congestive failure is past. To avoid straining at stools, constipation should be treated with laxatives or a retention enema as required.

5. Anticoagulant Therapy

Considerable difference of opinion exists at present concerning the use of anticoagulants in coronary occlusion.

The reduction in coagulability may be of benefit whether the cause is a coronary thrombosis, a sclerotic occlusion or a combination of both, since a tendency towards formation of parietal thrombi in the heart cavities is undoubtedly present (5). This means a potential risk of embolic incidents which are not seldom fatal. Furthermore, thrombosis in the pelvic and leg veins is also a potential danger, because of the prolonged bed rest necessary.

However, it is now felt by many that in *mild cases* of occlusion the use of anticoagulation and its subsequent hazards is unjustified, since the incidence of embolic phenomena is very small and accounts for no more than 1% of fatalities.

It is believed, therefore, that *anticoagulating drugs are indicated* only under the following conditions associated with coronary occlusion:

1. Signs of congestive failure
2. Severe or prolonged shock
3. Previous coronary occlusion
4. Significant cardiac enlargement
5. History or evidence of peripheral phlebitis, pulmonary, or peripheral embolism.
6. Serious cardiac arrhythmias: e.g. auricular fibrillation or flutter, bundle branch block, ventricular tachycardia.
7. Obese or debilitated patients whose movements are restricted and who may require extra-prolonged bed rest.

Contraindications to anti-coagulant therapy are:

1. Peptic ulceration or ulcerative colitis
2. Moderate to severe hypertension
3. Hemorrhagic diathesis
4. Lack of efficient laboratory facilities for prothrombin determinations, and the services of a blood bank.
5. History of hepatic or renal disease.

In the presence of one or more of the above indications, and the absence of any contraindication, *a suggested anticoagulating routine* is as follows:

1. Determine patient's normal prothrombin time (usually 10 to 15 seconds).
2. Heparin, 50 mg., q4h I.V., or as a continuous drip, 250 mg. over 24 hours. Heparin is continued during the first 24 to 48 hours until the action of dicoumarol becomes manifest.
3. Dicoumarol, 300 mg., p.o., given when heparin therapy is started; — followed the second day with 200 mg. and the third day with 100 mg.

During the first 2 weeks of treatment, prothrombin time determinations should be taken daily and maintained at 2 to 2½ times the control time, i.e. between 25 to 35 seconds. In most patients this result is achieved with a daily dose of 50 to 150 mg. of dicoumarol. Anticoagulant therapy is usually maintained 21 to 28 days.

The urine should be inspected grossly every day and urinalysis done every 2 to 3 days. If a significant number of red blood cells are found in the urine the

dosage should be reduced or the drug temporarily stopped. If the prothrombin time is prolonged to 60 seconds or more, or if bleeding occurs, the drug should be discontinued and the patient given intravenous Vitamin K_1 oxide, 50 mg. The emulsion of Vitamin K_1 oxide may also be given orally.

If hemorrhage occurs as a manifestation of heparin therapy, protamine sulphate, 1 mg. per mg. heparin given in the preceding 4 hours is rapidly effective in shortening the clotting time.

It is important to remember that anticoagulants are contraindicated in nonspecific pericarditis since they may cause pericardial hemorrhage. It has even been suggested that they be withheld in coronary occlusion when a definite pericardial friction rub is present, since hemopericardium with cardiac tamponade has been observed in several cases following anticoagulant therapy (7).

In no instance should oral anticoagulant therapy be instituted unless facilities are available for reliable prothrombin time determinations. Even if these are present, occasional mistakes are made and therefore the clinical picture should be closely observed.

COMPLICATIONS

1. Severe Shock

Coronary thrombosis is almost always accompanied by a certain amount of shock. It is important for this reason that blood pressure readings be taken at intervals, preferably every hour, or more frequently if the situation warrants it. If the systolic pressure falls below 100 mm. Hg. and remains there for any length of time, an attempt should be made to raise the pressure by means of drugs. Numerous drugs of the pressor amine group are available: norepinephrine, neosynephrine, paredrine hydrobromide, amphetamine, or mephentermine sulphate. The latter drug has the capacity to increase peripheral pressure without notably increasing cardiac irritability. Suggested intramuscular dosage of this drug is approximately 15 mg. repeated as necessary to maintain a pressure of 90 to 100 mm. Hg. Norepinephrine (Levophed) may be given slowly by the intravenous route (4 mg. in 1000 c.c. glucose in water). It may increase cardiac irritability to some extent and rarely may cause arrhythmias. Localized superficial gangrene as a result of intense vasoconstriction has been observed in some patients receiving the drug for long periods. Apart from the use of vasopressor drugs, a patient in shock should immediately be placed in a moderate Trendelenberg position and oxygen administered.

2. Congestive Failure

In cases displaying pulmonary congestion or other evidence of congestive failure, intravenous strophanthin or Cedilanid should be administered.

In some instances, clinical signs of heart failure may be absent, yet the venous pressure, if determined, is found to be elevated. Consequently, if a patient in shock does not respond to vasopressor drugs, it is worthwhile to administer digitalis.

3. Cardiac Arrhythmia

Arrhythmias of all types are frequently associated with coronary occlusion. The most common are premature beats and paroxysmal auricular fibrillation, flutter and tachycardia. They do not appear to alter the prognosis very significantly since they often are of brief duration and remit spontaneously. For this reason it is not necessary to begin specific treatment of these arrhythmias immediately if the patient is comfortable. Sedatives should be used liberally because the tachycardia is apt to cause anxiety. If the arrhythmia does not cease in ½ to 1 hour, specific therapy should be instituted. If there is any indications of incipient heart failure

or shock at the onset of the arrhythmia, treatment should be started promptly.

Ventricular tachycardia is a more serious and persistent complication in acute coronary occlusion. It usually responds to pronestyl or quinidine and these drugs should be given promptly.

4. Pulmonary Embolism

Pulmonary emboli usually originate in the lower extremities and lodge in the lower lobes. It is possible that a small percentage arise in the right auricle in the presence of atrial fibrillation or prolonged congestive failure.

The symptoms of pulmonary embolism are variable. There may be severe chest pain, dyspnea, and collapse simulating coronary occlusion, or merely cough, fever, or tachycardia. Pulmonary emboli usually do not occur until after the first week of the attack, most commonly during the second week. About 1/3 of the patients with pulmonary embolism expire within a few minutes or hours despite all treatment. If a patient survives more than 3 to 4 hours, he probably will recover(7). In some cases it is difficult to distinguish between pulmonary embolization and fresh myocardial infarction. The following points are helpful in making a differential diagnosis.

(1) Although fever, tachycardia, and chest pain may be present in both conditions, and cough may occasionally occur in cases with coronary occlusion and mild congestive failure, the presence of cough and hemoptysis favours the diagnosis of pulmonary embolism.

(2) Calf tenderness or a positive Homan's sign, viz. discomfort behind the knee on forced dorsiflexion of the foot, indicates the presence of a thrombotic process in a lower extremity and, therefore, possible pulmonary embolization.

(3) A loud pulmonic second sound, not previously heard, may indicate pulmonary hypertension secondary to infarction and the finding of a hazy patch of infiltration on chest X-ray may further suggest the diagnosis of an embolic episode.

The treatment is largely aimed at prophylaxis as discussed previously.

SUMMARY

The clinical recognition, confirmation, and management of acute myocardial infarction is discussed.

Although ischemic heart disease to some extent belongs to advanced age, it must be realized that it afflicts and kills a large number of individuals in their prime and at a stage of life when their contribution to family and community is invaluable.

Regardless of the ultimate outcome or severity of symptoms, coronary thrombosis is a matter of major importance which requires prompt action with the least possible confusion on the part of those persons charged with the responsibility of the patient.

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Management of Burns

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INTRODUCTION

Burns are caused by thermal, electrical, radioactive or chemical agents, and are the result of a coagulative destruction of the body's surface layers most often. However, the systemic effects associated with severe burns often offer a greater challenge in the management than do the local ones.

This paper deals mainly with the assessment and treatment of thermal burns and commonly associated complications. The pathologic physiology is briefly presented in order to aid in the understanding of local and systemic manifestations and early and late complications of burns which will be discussed subsequently.

PATHOLOGIC PHYSIOLOGY

Vasodilatation in the burn area associated with a marked increase in capillary permeability leads to extravasation of plasma into the interstitial spaces, causing a detectable hemoconcentration within a few hours. The edema reaches a maximum in 36 to 42 hours.

Local heat destruction of red blood cells tends to cause an anemia which may be masked initially by the hemoconcentration. Factors which add to the anemia later are increased fragility of red blood cells, reduced synthesis of hemoglobin and a bleeding burn wound.

It can be easily understood how the above factors, viz., plasma loss and hemolysis, along with electrolyte disturbances may readily precipitate severe secondary shock which may prove rapidly fatal in a severely burned person without proper therapy.

ASSESSMENT

In order to formulate a satisfactory method of treatment it is necessary to assess as accurately as possible the burn depth and the extent of the area involved.

Classification of Depth

First Degree

There is simple erythema, warmth, tenderness, pain, edema, but no vesiculation. It heals in a few days.

Second Degree

These burns are sometimes divided into superficial and deep. There is partial destruction of the dermis but without destruction of all deep epithelial cells. There may be small blebs, some broken, some intact with an external ooze of plasma, visible pink corium and minimal subcutaneous edema. It may heal in 2 to 3 weeks. Regeneration takes place from remaining hair follicles, sweat and sebaceous glands.

Third Degree

This is full thickness destruction of the skin. It may have a dry dead white, brown or charred appearance, and fat or coagulated muscle may be seen. An eschar forms which sloughs leaving an ulcer. Epithelial regeneration takes place from the margins, and severe contractures may ensue if the wound is not grafted early.

Burns intermediate between the above cannot be classified until epithelialization

or slough have become evident. Moderate hypoalgesia in partial thickness burns and marked hypoalgesia or anesthesia in full thickness burns may be noted in response to a pin prick. Hemoglobinemia and hemoglobinuria denote deep extensive burns.

Estimation of Area Involved

This may be determined by using Berkow's formula (with Lund's modification for infants and children) or the "Rules of Nine" proposed by Wallace. In the latter the head and neck and each upper extremity count as 9% each of the body area, while anterior trunk, posterior trunk and each lower extremity count as 18% each of the body area. The genitalia and perineum are considered 1% each.

TREATMENT OF BURNS AND THEIR COMPLICATIONS

Prophylaxis

Prophylaxis as regards burns is of huge scope, encompassing many aspects of industrial and home safety. The multitude of chinks in our armour against burn accidents is readily realized by many health and safety organizations who are moving steadily toward improvement of this situation. In the meanwhile it is necessary to attempt rescue of the many burn victims which result.

First Aid

Minor Burns

The parts should be cleansed gently and sterile gauze dressings gently applied.

Serious Burns

The burned area should be wrapped in sterile or at least clean dressings and the patient transported to hospital immediately. Morphine, grains 1/6 to 1/4, may be given subcutaneously or slowly into a vein if indicated.

A. First, Second and Third Degree Burns Involving Less Than 15% of the Body Surface (in an Adult)

1. Early Definitive Treatment

These may all be treated in a similar manner with minor variations based on common sense.

Ideally, when the patient arrives in the emergency room of the hospital he should be placed on a sterile sheet after his clothing and first aid dressings have been removed by gowned and masked attendants. He is then covered with another sterile sheet. The area and depth of burn should be carefully estimated and recorded. An estimate of his weight should also be made.

2. Surface Treatment

Compression Dressing Technique

This technique is based on the teaching of Koch, Mason and Allen, who stressed that burns are analagous to other traumatic open lesions and should be treated as such. A culture should first be taken from the burned area. Gentle cleansing with a bland soap, or detergent with hexachlorophene, rinsing with normal saline, application of a non-adherent compression dressing and splinting converts a burn from an open contaminated wound to a closed clean wound as rapidly as possible. Fine mesh gauze, dry or impregnated lightly with petrolatum is applied directly over the burn. Over this, sterile absorbent padding is placed evenly, thickly and loosely, and secured by a wide stretchable bandage. Elevation may help prevent stasis and reduce swelling. The dressing must be kept dry on the outside to prevent possible contamination of the wound.

Exposure Technique

This technique had in recent years been restricted to burns of the face and perineum until the work of Wallace in 1948, who extended the treatment to other

regions. It appears to be the most practicable way of dealing with mass burn casualties at the time of a civilian or military disaster. It is based on the principle that bacterial growth is inhibited by a cool dry surface. In circumferential burns of the trunk complete exposure may be impossible.

The burned and surrounding areas are cleansed as before and rinsed with saline. The patient is placed in a position giving maximal exposure, and the affected parts are immobilized. Exudate of a partial or full thickness burn usually dries in 48 to 72 hours and forms a crust which tends to be softer with the application of aluminum powder. This has less cracking tendency and therefore less chance of secondary infection.

3. Systemic Treatment

Pain can be treated with codeine and acetylsalicylic acid. More severe cases may require morphine subcutaneously or slowly by the intravenous route. Morphine, however, is contraindicated if a patient is intoxicated, or in shock, or suffering from carbon monoxide exposure or respiratory tract damage. These complications which will be discussed later, suggest instead the use of demerol.

Exposure Technique versus Occlusive Technique

The results obtained with either of these techniques depend more on the proper application by trained personnel rather than apparent advantages or disadvantages which appear from an academic point of view, to be inherent in one or other method.

Temperature

Whereas the occlusive technique would appear to protect from cold there is the disadvantage of possible hyperthermia when a large portion of the body is covered.

Infection

Investigations have shown there tends to be less infection with the exposure technique when it is carried out properly. Contamination is prevented in the pressure dressing method only so long as the exterior of the dressing remains dry.

Fluid Loss

The amount of fluid lost by extravasation in the burn area is only slightly reduced by a dressing. In the exposure method plasma loss is minimal once the crust forms within 48 to 72 hours.

Also, in the exposure technique there appears to be less odour and fever, and smaller doses of narcotics and antibiotics are required. Respiratory movement of the thorax and abdomen is not restricted.

B. Second and Third Degree Burns Involving More Than 15% of the Body Surface (in an Adult)

Early Definitive Treatment

Both *surface* and *systemic* treatment is much the same as that mentioned for second and third degree burns covering less than 15% of the body surface, but there are a few very important differences which are discussed below as clearly complications.

EARLY COMPLICATIONS

1. Secondary Shock

This is undoubtedly the most important of the early complications, for without early and vigorous therapy death will ensue. Shock is the result of a dimunition of blood volume (chiefly the plasma fraction), disturbance in electrolytes, plasma proteins, and various biochemical and enzyme systems, and can be anticipated in burns greater than 15% in an adult. It is important to recognize and treat impending shock early rather than make an easy diagnosis in the full blown stages. Impending shock is characterized by pro-

gressive hemoconcentration, falling plasma protein concentration, falling blood pressure, rising pulse and a lowering in urinary output.

When the patient is first seen, a suitable vein should be cannulated immediately and blood drawn for determinations of hemoglobin, hematocrit, blood type and cross-match. Procedures which tend to increase shock, such as dressing, should be avoided until shock is prevented or treated.

Replacement Therapy

The amount and rate of replacement depends on the following:

(1) Area of surface burned. However, face and genitalia which are more vascular tend to have higher fluid losses.

(2) The fluid equilibrium. The rate of fluid loss is maximal in the first few hours and gradually decreases so that an equilibrium is reached in 36 to 48 hours, i.e., fluid lost equals the amount returned to the circulation from the interstitial spaces.

(3) Clinical and laboratory data.

(4) Age and general condition of the patient.

The best guides are the general clinical appearance of the patient and the urinary output. Therefore an accurate hour-to-hour record of urinary output with an in-lying catheter is necessary for the first 24 to 48 hours.

Therapy should be individualized, but a number of formulas have been devised as *guides*. The Evans formula is the most widely used. It states that 1 ml. of colloid (whole blood, plasma, or plasma expander) per kilogram of body weight per per cent of body area burned is given intravenously during the first day and one half this amount the second day. Equal amounts of isotonic saline are given, and also for an adult, 2000 ml. of 5% glucose in water on each of the first two days. If the burn is deep and extensive, half the colloid requirement is given as blood. Depending on the severity of the burn and circulatory factors, the saline and glucose solutions are given intravenously or orally.

An upper limit is placed on the amount of fluids given. This is the quantity indicated by the formula for a 50% burn.

One half the fluid requirement for the first day is given in the first four to six hours, one quarter in the next six hours and one quarter in the next twelve hours. The rate of administration is sufficient to maintain the hemoglobin level below 19 grams and the urinary output at about 25 to 50 ml. per hour for an adult. Minimum urinary output (25 ml./hr.) through limitation of colloid and salt administration should be attempted in patients with respiratory injury or serious cardiac disease.

In a previously normal adult with severe shock and an extensive burn, as much as 2000 ml. may be given in an hour without danger of overloading the circulation.

Vitamins

Experimental work indicates depletion of water soluble vitamins during burn shock as well as traumatic shock. It seems logical therefore to administer large doses of B-complex vitamins and ascorbic acid early in the case.

Position

Raising the foot of the bed improves cerebral circulation in the first few minutes when effective shock therapy is being instituted.

Environmental Temperature

Extremes of temperature are detrimental. Optimal environmental temperature is 24 degrees centigrade. If the rectal temperature is below 34 degrees or above 40 degrees, measures must be taken to warm or cool the patient promptly.

Oxygen

This is only indicated when there is accompanying respiratory tract injury.

Pressor Drugs

These are of no value and may be harmful.

Sedation

Morphine and other sedatives must be used cautiously.

Adrenal Cortical Extracts

There is no evidence of beneficial effects. Rarely it may help a patient with adrenal exhaustion.

2. Kidney Injury

The changes which may occur in kidney function and morphology are similar to those in lower nephron nephrosis.

Etiology

The cause would appear to be reduced renal blood flow and anoxemia secondary to impairment of the general circulation, acidosis, methemoglobinemia and methemoglobinuria.

Prophylaxis

These changes may be averted by combating shock adequately and vigorously. Prompt and continued alkalinization of urine during the period of methemoglobinuria (usually 48 to 72 hours) may be important. This may be done initially by an intravenous injection of sodium bicarbonate. Input-output records should be kept for at least one week in patients with burns over 10% in body area. The output should be maintained at 1000 to 15000 ml. daily. Too much fluids and electrolytes must be avoided in the period of reabsorption of edema fluid which begins after 36 to 48 hours.

Blood protein, N. P. N., plasma bicarbonate and chloride, and routine urine determinations should be done frequently.

3. Respiratory Tract Injury

Pathology

The fundamental pathologic lesion is a laryngotracheobronchitis which may go on to necrotization.

Symptoms

Symptoms may include sore throat, cough, hoarseness, dyspnea, cyanosis, wheezing, stridor, sputum production (mucoid, purulent or bloody), and restlessness.

Signs

These may include areas of dullness, hyperresonance and rales of all varieties. An X-ray may show areas of atelectasis, emphysema, congestion and consolidation. Respiratory tract injury should be suspected in a case with face burns or a history of loss of consciousness or inhalation of large quantities of smoke.

Etiology

This type of injury tends to occur with inhalation of smoke and carbonized particles and occasionally toxic fumes.

Treatment

The room should be humidified and a tracheotomy set kept ready for use in case of laryngotracheobronchial spasm, air hunger or obstruction. Persons in contact with the patient should be masked. The patient should be turned frequently and encouraged to cough and respiration should be checked every half hour.

If hoarseness, dyspnea or stridor occurs, an oxygen tent may be used (10% carbon dioxide every one to two hours encourages deep respiration). With bronchial spasm, aminophylline intravenously can be given. Restlessness is treated with more oxygen. If there is pain, demerol is given; if nervousness, barbiturates.

Antibiotics, if necessary, are varied depending on the results of cultures.

If intravenous solutions are being given, especially electrolytes, it is important to watch for signs of pulmonary edema.

4. C. N. S. Disturbances

These may be indicated by delerium, stupor, coma, convulsions, hyperpyrexia, and Cheyne-Stokes respiration in extensive burns. The cause is uncertain. It may be secondary effects from a generalized edema, or a circulatory toxin.

5. Hyperpyrexia

Frequently in adults with extensive burns and children with lesser burns, the temperature may go above 41 degrees centigrade. The oral temperature may be misleadingly low, therefore, the rectal temperature should be taken. If the temperature is over 41 degrees in a burn patient more than a very few hours death is almost certain.

Treatment consists of ice water sponging to all exposed skin. They are sponged until the rectal temperature is maintained at 39 degrees centigrade. External cooling blankets, etc., may be used.

6. G. I. Tract Ulcers

These were first recognized by Curling in 1842. They occur most commonly in the esophagus, stomach, and duodenum, but on the whole are relatively uncommon complications. When they occur, however, they are extremely serious. Symptoms include epigastric pain, hematemesis and melena.

7. Emotional Disturbances

Contributing factors may be poor nutrition, and regular use of sedatives and narcotics.

LATER TREATMENT OF BURNS

As previously mentioned, in the occlusive dressing technique the original dressing is left in place for ten days to two weeks except for very superficial second degree burns which are probably healed by this time. For deeper and more extensive burns, when it is time to change dressings, it should be done under stringent aseptic conditions, preferably in the operating room. The patient is prepared with a mild sedative and cyclopropane is the anesthetic of choice. The status of the burn wound is appraised and further needs estimated. Deep second, mixed, and third degree burns are still covered with slough.

Slough separates spontaneously in 3 to 6 weeks, more rapidly in vascular areas such as the face. However, to avoid slow healing and disfiguring scars, third degree and certain deep second degree burns of more than about two centimeters diameter require skin grafting.

Immediate excision and grafting is theoretically sound since the area would be healed in a week or ten days. Delayed excision and grafting at 10 to 14 days is commoner, however, since the general condition of the patient has stabilized and it is possible also to distinguish partial-thickness from full-thickness burns. Immediate excision and skin grafting is indicated only in *obviously deep* burns of a relatively small area.

Excision and grafting in 10 to 14 days may be carried out in the following manner.

A general anesthetic, preferably cyclopropane is used. There is an edematous layer between viable and non-viable tissues through which the excision takes place. In this way the necrotic slough is excised with minimal bleeding. With extensive burns surgical excision will produce shock, therefore, whole blood should always be administered. The excision may be done in stages if the patient is in critical condition.

Removal of necrotic slough decreases the patient's temperature and white blood count, and the general condition improves. After excision of an area too large for

immediate coverage with grafting, the area is redressed. The patient is brought back to the operating room 5 days later and the grafting is then performed.

Though autografts are certainly best, homografts have a definite application, particularly in children with a wide burn area. With granulating areas covered, though only temporarily, the general condition can be made to improve by the time the grafts dissolve. Autografting can then be successfully carried out.

Later on, the use of physiotherapy hastens rehabilitation.

LATE COMPLICATIONS

1. Infection

This interferes with local healing and intensifies the general physiologic and biochemical derangement associated with burns. Clark and Colebrook emphasize that the majority of infections in burns are "added infections", that is, infection of burned surfaces with organisms not previously present.

Prophylaxis

(1) Masking, gowning and scrubbing of all personnel in contact with an uncovered burn.

(2) Keeping exterior of dressings used in occlusive dressing technique dry.

(3) Segregation of infected cases.

(4) Exclusion of attendents and visitors with colds.

(5) Immobilization of burned and adjacent areas.

(6) Maintenance of dry wounds.

(7) Removal of dead tissue of deep burns and grafting as soon as possible.

(8) Maintenance of nutritive and general condition of the patient.

(9) Prophylactic immunization against tetanus (toxoid or antitoxin as indicated).

(10) Antibiotics. These should not be administered to all burn patients. They should be restricted to patients with deep partial thickness and full thickness burns and patients with respiratory tract injury. Penicillin (unless allergy) is usually started. Later this may be varied depending on culture.

2. Scars and Contractures

These are prevented by early excision of slough and skin grafting, control of infection, and intelligent physiotherapy.

3. Anemia

Hemoglobinuria and hemoglobinemia are observed immediately after extensive deep burns and continues for about 72 hours. It is estimated that 10% of red blood cells are hemolyzed during this time. Chronic anemia may result from loss of blood in dressings, especially in patients in a poor nutritional state where granulations are poor and edematous. Also, hemoglobin and red blood cell formation may be depressed.

It is important to keep the hemoglobin concentration at 85% or above. This may require 1000 ml. of blood per week in a patient with deep extensive burns.

4. Nutritional Disturbances

Malnutrition results in decreased resistance to shock and infection, delayed wound healing including failure of skin grafting, impaired gastro-intestinal and liver function, prolonged convalescence and increased mortality.

A severely burned male (over 20% third degree) would appear to require a daily intake of about 4 grams of protein and about 55 to 60 calories per kilogram. Too high carbohydrate intake too early may cause serious hyperglycemia, glycosuria and dehydration. This can be alleviated by substituting fat for some of the carbohydrate.

Vitamins, water soluble vitamins especially, are required in larger amounts.

The oral route for feeding is the most satisfactory and the earlier the better. Smaller amounts should be given at first to prevent gastro-intestinal upsets.

SUMMARY

Thermal burns were discussed under the headings of pathologic physiology, assessment, treatment, and early and late complications. The relative merits of both the compression dressing technique and exposure technique were outlined. Under later treatment of burns, skin grafting was dealt with briefly. Complications which were presented included secondary shock, kidney injury, respiratory tract injury, infection, contractures, anemia, and nutritional disturbances.

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"In medicine everything, or nearly so, depends upon the acuteness of perception . . . Certainty is found to exist in the sensations of the artist himself, rather than in the principles of the art. For studying the phenomena which living bodies exhibit to our view and for tracing their history with accuracy it is not necessary that we know the nature of the principle which animates them, or the manner in which it brings their powers into action . . . observation and experiment and reasoning are sufficient for our purpose—we require nothing more."

Cabanis, *Du Degré de Certitude de la Médecine,* Paris, 1803.

"Has anyone at the end of the 19th century any distinct notion of what poets of a stronger age understood by the word 'inspiration'. If not I will describe it. If one had the smallest vestige of superstition left in one, it would hardly be possible completely to set aside the idea that one is the mere incarnation, mouthpiece, or medium of an almighty power. The idea of revelation, in the sense that something which profoundly convulses and upsets one becomes suddenly visible and audible with indescribable certainty and accuracy, describes the simple fact. One hears—one does seek; one takes—one does not ask who gives; a thought suddenly flashes up like lightning, it comes with necessity, without faltering—I have never had any choice in the matter."

Friedrich Nietzche, *Ecce Homo,* p 101, 1882.

The Differential Diagnosis of the Acute Abdomen

KATHLEEN SANDOR, '58

INTRODUCTION

It may be said that in no other symptom complex in the entire field of medicine must the physician lean so heavily on the information provided by history and physical examination, as in the differential diagnosis of the acute abdomen.

The importance of an honest attempt to make an accurate diagnosis and the necessity for speed cannot be emphasized too strongly. The surgeon, aware of the excellence and relative safety of modern operative techniques may, at times, be too eager to open the abdomen with only a perfunctory examination and long experience to assure himself that the patient will ultimately come to surgery. A painstaking attempt at diagnosis is vitally important, since only in this way can the present knowledge of the acute abdomen be increased. Few conditions offer such an opportunity to correlate symptoms with pathology in the living. Apparent improvement in the patient's condition should never encourage the physician to delay in making a diagnosis.

A list of the various pathological conditions causing acute abdominal emergencies is readily available in any good text-book of surgery or differential diagnosis. It is not the purpose of this paper to enter into a discussion of these entities; rather, since history and physical examination are so fundamental to the diagnosis of the condition producing the acute abdomen, to offer a method for differential diagnosis that is as complete as space permits.

ESSENTIALS OF DIAGNOSIS

1. Attempt to Make a Diagnosis

It seems redundant to state that in order to make a diagnosis, one must attempt to make a diagnosis. It was pointed out previously, however, that seldom is the physician so prone to rush an obviously ill patient to operation with no sincere attempt to pin-point the cause of the patient's acute abdomen.

2. Speed in Making a Diagnosis

The necessity for this should be obvious. Acute abdominal conditions are always potentially fatal. The outline which will follow as a suggested method of diagnosis may appear to embody the antithesis of speed, but increasing experience yields speed and skill. Because speed is so essential, it should be an invariable rule never to allow morphine to be administered until a diagnosis has been made. Relief of pain provided by morphine is too prone to provide both physician and patient with a false sense of security.

3. Apply Knowledge of Anatomy and Physiology

Diagnostic aid provided by keeping anatomical details in mind is best exemplified by those structures whose positions are the least variable, e.g. muscles and nerves. Intra-abdominal inflammation can often be localized with great accuracy by remembering that muscles adjacent to the inflammatory process, will be irritated, and pain is, therefore, elicited on movement of these muscles. This principle is applied in the psoas and obturator tests.

It should also be borne in mind that subphrenic abscess will produce pain on movement of the diaphragm.

Exact knowledge of the course and distribution of the spinal nerves is invaluable. Illustrative of this is the pain around the umbilicus that occurs early in acute appendicitis. Both the appendix and the skin around the umbilicus are supplied by the 10th thoracic nerve. Another pain of segmental distribution of great significance is that referred from the diaphragm to the shoulder region.

Physiology is as important in the localization of obstructive lesions as is anatomy in the inflammatory lesions. Pain from a hollow viscus is usually due to stretching of the viscus. Thus, normal peristaltic waves become acutely painful in a patient as they attempt to overcome an obstruction. Distension above the lesion with fluid and gas adds to the distress.

4. Eliminate Medical Diseases as Possible Causes of Abdominal Pain

A thorough physical examination should *never* be neglected in any case. More than one surgeon has been guilty of sending a patient to operation ignorant of the presence of albuminuria or tabes dorsalis.

METHOD OF DIAGNOSIS

History

A careful history is a virtual necessity. If the patient is not sure of his facts, his family, if available, must be questioned. All the following points should be carefully investigated:

1. *Age*

Certain diseases are much more common in certain age groups. If the patient is a child it is well to remember that acute intussusception is more common in infants under two years. Appendicitis is a common cause of acute pain in adolescents, whereas acute pancreatitis is rare under middle age. Females in the reproductive period should be carefully examined for conditions due to derangement of the developing ovum. Should the case be one of intestinal obstruction, a cancerous cause for the obstruction is most common over forty. The commonest cause of right lower quadrant pain in a patient over sixty with a previous appendectomy is a perforated caecum due to carcinoma.

2. *Exact Time of Onset*

This may be very important, not only as a help in diagnosis, but also as a means of differentiation between real and imaginary pain. If the patient definitely states that he was awakened by pain, the condition is very often serious. Acute appendicitis commonly, and perforated ulcer fairly commonly onset by awakening the patient out of sleep.

3. *Mode of Onset*

Slight exertion may precipitate an acute condition; a very strong aperient may precipitate or worsen an acute condition. This is not infrequently true of appendicitis. A drinking bout may precede acute pancreatitis or the perforation of a peptic ulcer.

A very acute onset with fainting or collapse is common with perforation of an ulcer or rupture of a tubal pregnancy. Gradual onset is typical of intestinal obstruction, while strangulation of the bowel begins very acutely.

4. *Pain*

Pain is the very essence of the acute abdomen. No aspect should be overlooked. Enquire carefully about:

i. Situation of the Pain at Onset.

Pain felt over the entire abdomen at once is frequently caused by rupture of a viscus and flooding of the peritoneal cavity with blood or irritating fluid. A point of maximum tenderness may give the clue to the lesion, e.g. tenderness over

the entire abdomen with a maximum point in the right hypochondrium is likely caused by a perforated duodenal ulcer. Small intestine pain is felt in the epigastric and umbilical areas. Large gut pain is usually felt in the hypogastrium.

ii. Shifting or Localization of the Pain

This may well be an extremely significant point. This history is particularly typical of acute appendicitis; the pain onsets in the epigastrium and localizes in the right iliac fossa. Keep in mind, however, that occasionally the same sequence is seen with a perforated peptic ulcer. Other points in the history and examination must be relied upon to make the diagnosis.

iii. Character of the Pain

This is always worth investigating, but the patient may be unable to describe the pain in precise terms that will be meaningful to the physician. A typical story suggestive of one of the following may, however, be helpful.

a. Perforated gastric ulcer—acute burning pain; associated peritonitis will make any movement painful, therefore, the patient lies very still.

b. Acute pancreatitis—agonizing steady pain.

c. Biliary colic—sharp constricting pain; the patient will writhe in bed with the intermittent attacks.

d. Intestinal obstruction—recurrent attacks of griping pain.

iv. Radiation of the Pain.

Radiation is frequently a diagnostic sign. This is especially true of the colics —the pain radiates to the area of distribution of the nerves from that segment of the spinal cord which supplies the affected part. The pain of biliary colic is referred to a point below the inferior angle of the right scapula. Acute lesions of the upper abdomen and lower thorax will have pain referred to the top of the shoulder on the same side as the lesion. Pain may be influenced by respiration. Peritonitis or intraperitoneal abscess near the diaphragm will cause pain on inspiration.

v. Pain during micturition may be due to pyelitis, a renal or ureteral calculus, a pelvic abscess lying close to the bladder or an inflamed appendix close to the right ureter.

5. *Vomiting*

Except for acute gastritis, vomiting has three possible causes.

i. Severe irritation of the nerves of the peritoneum or mesentery, e.g. due to irritating fluids poured into the peritoneal cavity when an ulcer or an inflamed appendix perforates. In this type, the patient vomits very soon after the irritation onsets, but dilution of the irritant by fluid from the peritoneum lessens the vomiting.

ii. Obstruction of Involuntary Muscular Tube.

In the colics, biliary, renal, intestinal and uterine, the tube behind the obstruction becomes dilated and each peristaltic wave dilates the muscular wall still further, so that the pain comes in spasms, with vomiting at the height of the spasm. Holding up of the intestinal contents and antiperistalsis are additional factors causing vomiting in intestinal obstruction.

iii. Action of absorbed toxins upon the medullary centres, e.g. septic peritonitis.

The relationship of pain to vomiting is a point of interest. Sudden severe stimulation of the peritoneum and acute obstruction of the ureter or bile duct cause violent vomiting very soon after the onset of pain. In intestinal obstruction, the length of time after the pain before vomiting begins is some indication of the level of obstruction; vomiting is very late and is not a prominent feature of large bowel obstruction. Pain of acute appen-

dicitis *always* precedes vomiting, usually by three to four hours.

The frequency of vomiting varies directly as the acuteness of the condition. For this reason, vomiting at the onset of an attack of appendicitis may mean imminent perforation.

Vomiting is slight or absent with a ruptured ectopic pregnancy, large bowel obstruction, and intussusception.

It is usually wise to enquire into the character of the vomitus. This is seldom diagnostic except in long standing intestinal obstruction where the vomitus has progressed from gastric contents to the faeculant material in the lower ileum. Note that acute torsion of a viscus usually involves much retching with little vomitus. Nausea and loss of appetite may be considered different degrees of vomiting in a person who needs a greater stimulus to vomit.

6. *Bowels*

Bowel habits are not significant unless a definite change from normal bowel habits has occurred. Look for obvious blood or slime in the faeces and enquire about tarry stools. Cases of intestinal obstruction may well have a bowel movement after obstruction has occurred, due to emptying of the segment of bowel distal to the obstruction.

7. *Menstruation*

Never forget a complete menstrual history in a woman in the reproductive period of life. A missed period followed by a painful period, if pain is not usual, may indicate a threatening abortion or a tubal gestation. Abdominal pain in a woman fourteen days before the next expected period should always suggest mittelschmerz.

8. *Past History*

Particularly important are previous stories of appendicitis, ulcer, gall stones, jaundice, melena, hematemesis, and hematuria. Previous surgery may provide a clue, e.g. biliary tract surgery predisposes to acute pancreatitis.

Examination of the Patient

A meticulous examination is mandatory if a diagnosis is to be made. *Always* guard against the possibility of subjecting a patient to an unnecessary operation.

1. *General Appearance*

When the patient is pale, livid, and sweating, consider a perforated ulcer, acute pancreatitis, and acute strangulation of gut. Pallor and gasping respirations are quite typical of a severe internal hemorrhage.

A motionless patient with drawn up knees is quite likely to have severe peritonitis. Restlessness is typical of the colics or intraperitoneal hemorrhage.

If the patient looks reasonably well, keep in mind that recovery from initial shock does occur.

2. *Pulse*

A normal pulse does *not* indicate a normal abdomen, or mild disease. In the initial stages of the acute abdominal catastrophies the pulse is weak and rapid, but it often becomes normal later. A rapid pulse in advanced stages of peritonitis or haemorrhage is a very grave prognostic sign.

3. *Respiratory Rate*

If the rate is doubled, examine the chest carefully for pneumonia.

4. *Temperature*

There is nothing typical about the temperature in acute abdominal disease. With severe shock, toxaemia, or intestinal obstruction, the temperature is usually subnormal. Cases of early appendicitis will show no change in temperature; later a rise to 100-101°F. usually occurs. A high

fever is rare with acute abdominal conditions, and, if present, a thoracic or kidney infection should be looked for.

5. *Tongue*

Examination of the tongue is not too significant except in the diagnosis of uremia, which may simulate intestinal obstruction. The tongue in uremia is dry and heavily coated with brownish fur.

6. *Abdominal Examination*

A. Inspection

Note any local or general distension, evidence of herniae at the hernial orifices or femoral canal. Limitation of respiratory movements indicates rigidity of the diaphragm or abdominal muscles. Peristalsis should be watched for: it is not invariably present in cases of intestinal obstruction but is diagnostic when present. A drop of ether dropped onto the skin of the abdomen may initiate peristalsis. Abdominal distension in cases of intestinal obstruction may show the characteristic ladder pattern of small bowel obstruction, the distension being principally central in the abdomen; distension does not occur early in small intestinal obstruction. Early distension occurs in large bowel obstruction and the brunt of the distension is usually borne by the caecum; distension of the large bowel is usually seen toward the periphery of the abdomen.

B. Palpation

i. Muscular rigidity may be board-like and continuous, as with a perforated ulcer, or it may occur only when the fingers are pressed in. Do not confuse the voluntary contraction of abdominal muscles against the examining hand, with true rigidity. Rigidity is often absent in pelvic lesions or intestinal obstruction.

If rigidity can be broken down by steady pressure, the pain over an intrabdominal lesion will be increased, but pain originating in the thorax will be unchanged. Rigidity may be slight in patients with weak, flabby abdominal muscles, obesity or severe toxaemia in which reflexes are dulled. Deep pressure exerted over the site of the pain with sudden release may cause sharp pain. This is known as rebound tenderness and indicates inflammation of the parietal peritoneum.

ii. Hyperasthesia

Hyperasthesia is best detected with a light stroke of a point of a pin. It may be detected in the segmental distribution of that part of the spinal cord from which nerves supplying the diseased viscus come, or along the distribution of any peripheral nerve whose terminals are irritated by inflammation. It is most commonly found in appendicitis and usually indicates irritation of the visceral or parietal peritoneum.

iii. Palpation of the Loins

Resistance and tenderness in the loins indicate irritation of the quadratus lumborum by inflammation nearby, possibly a periphrenic abscess, inflamed kidney or retrocaecal inflamed appendix.

iv. Liver dullness is best detected normally in the right vertical nipple line from the fifth rib below the costal margin. A resonant note from the normal dull area denotes free air in the peritoneal cavity, due usually to rupture of the intestine or stomach.

C. Auscultation

The absence of normal bowel sounds indicates ileus or peritonitis. The very loud rumbling noises that may even be heard a distance away from the patient (i.e. borborygmi) suggest intestinal obstruction. It has been found that in the presence of sounds of intestinal movement injury to the intestine is very unlikely. Prolonged listening is necessary before one can say with certainty that movements are absent.

7. *Pelvic Examination*

i. Careful palpation above Poupart's ligament may reveal deep tenderness or a tumour mass.

ii. Digital rectal examination is very, very important. Under *no* circumstances should it be omitted. Press upwards, forwards, backwards and laterally, noting any mass and or tenderness in each area. In the male rectal examination may reveal an enlarged prostrate and a distended bladder. In the female swellings in the pouch of Douglas may be discovered and in this way an ectopic pregnancy or salpingitis may be diagnosed. Enlargement of the uterus may suggest pregnancy. Lateral tenderness on the right side may suggest appendicitis.

Abnormalities of the rectum itself may suggest a diagnosis; for example, strictures of the rectum due to fibrosis or carcinoma may be the cause of intestinal obstruction. In infants, the apex of an intussusception may be felt. Note any blood, slime, or pus on the examining finger when withdrawn.

8. *Special Signs, Helpful in Acute Abdominal Disease*

i. Ilio-psoas Rigidity

If this is extreme, the thigh on the side of the disease is flexed; in less severe cases, minor degrees of rigidity may be detected by extending the thigh on the affected side to the fullest extent. Reflex or direct irritation of the psoas muscle is the cause of pain on this maneuver.

ii. Obturator Test

The flexed thigh is rotated internally. Pain occurs with this test if the obturator internus muscle is irritated, through contact with an inflammatory mass such as an inflamed appendix or pelvis abscess.

iii. Rovsing's Sign

Pressure over the descending colon forces gas into the caecum or appendix causing pain in the region of the caecum if the appendix is inflamed. This sign is not, by any means, invariable, but it is helpful when present.

iv. Dance's Sign

The right lower quadrant appears to be empty in cases of caecal-colic intussusception.

v. Masses in the Abdomen

Movable masses enlarging migrate toward the midline in the lower half of the abdomen and laterally in the upper half.

vi. The release sign or rebound tenderness has been mentioned before.

9. *Chest*

Cover the chest thoroughly, to rule out pleurisy, pneumonia or cardiac disease as possible causes of acute abdominal pain. An E.C.G. may be necessary to make a certain diagnosis of the latter.

10. *Knee Jerks and Pupils*

If the pupils do not react to light or if even one knee jerk is absent, look for tabes dorsalis. Should a diagnosis of tabes be made with certainty, however, do not forget that a tabetic may still have acute abdominal disease. This may present an extremely difficult problem in diagnosis and a laparotomy may be necessary.

11. *Blood Pressure*

Low blood pressure will occur in internal haemorrhage, shock or circulatory failure following intestinal obstruction. It is important not to neglect blood pressure readings in a case of abdominal pain, since low blood pressure will not admit any delay in diagnosing and treating the condition.

Radiological Examination

This is, indeed, a valuable adjunct to physical examination, but the physician should be cautioned against short-cuts in his examination in the belief that X-rays will give the final diagnosis.

Ideally, no water enema should be given prior to examination, since fluid levels remaining in the intestine three to four hours afterwards may confuse the picture. Films of at least three positions should be obtained. Supine, upright and left lateral recumbent are the positions most usually examined.

The following non-specific evidences of intra-abdominal pathology should be sought.

1. Normally the contours of the liver, spleen, and kidneys are visible, but in acute abdominal conditions they may be blurred due to fluid, pus, or blood within the abdominal cavity.

2. On the posterior abdominal wall the psoas shadow is obliterated in abdominal disease due to contraction of the muscle. The flank may be contracted, showing on X-ray as a medial bulge. The spine may be curved laterally and if this is so, the concavity is toward the side of the lesion. These three signs are most commonly seen together with perforated duodenal ulcer, appendicitis, and ureteral calculi.

3. The small bowel is normally free of gas except in children not yet walking. Therefore, gas in the small intestine is indicative of intestinal obstruction and the more distal the obstruction, the more gas collects. Minimal gas collects in strangulating obstructions.

4. Fluid in the peritoneal cavity in small amounts is most easily detected in the lesser pelvis after the patient has been sitting up. Increased amounts collect along the border of the flank and between the intestinal loops. In cases of ascites, the entire abdominal field is covered by a diffuse density with poor outlining of the parenchymatous organs.

5. Pneumoperitoneum is usually due to a ruptured ulcer—70 to 80% of perforated ulcer cases show pneumoperitoneum. When the patient is sitting or standing, gas is seen beneath the diaphragmatic domes.

Special Pathological Findings

1. Intestinal Obstruction

If the occlusion is low in the small intestine there are several characteristic findings on X-ray: fluid levels in the distended loops, the "poker-chip" pattern of jejunal mucosal folds, and the "hair-pin" turns of the distended loops.

Large bowel obstruction shows up on a plain film of the abdomen as a ballooning of the colon above the obstruction. The small bowel may be secondarily dilated.

2. A volvulus usually can be seen as a dense mass on a plain film.

3. Local Inflammation

An abscess is seen as a local opacity, surrounded or delineated by gas-filled loops of bowel with oedematous loops of mucosa. The commonest sites are the flanks, subdiaphragmatic, pericaecal and the lesser pelvis.

Laboratory Examination

Although time is at a premium, and only short tests are possible, a fairly complete blood study should be done. A haemoglobin estimation if low will suggest chronic bleeding. A haematocrit is important to indicate the degree of dehydration. To complete the blood picture a white blood count is done. At the same time grouping and cross-matching of the patient's blood is done if indicated.

A complete urinalysis is important. Red cells in the urine may confirm suspicion of ureteral calculi. Pus in the urine may suggest renal or bladder infection as the source of the patient's pain. Bile or bile pigments in the urine should be looked for. A urine sugar is very important since patients with acute abdominal disease are prone to come to surgery. Diabetes will influence the handling of the patient at operation. If acute pancreatitis is suspected, a serum amylase level may be helpful.

Remember, however, that too great dependence on laboratory examinations may be harmful; for example, the white count may not be elevated in appendicitis until the appendix perforates. The serum amylase level may have returned to normal if a case of acute pancreatitis is not seen until 48 hours after onset. This test may be very unreliable unless consideration is given to other conditions which may produce elevated amylase levels, e.g. spasm of the sphincter of Oddi, perforation of a posterior duodenal ulcer, inflammation or obstruction of the salivary glands or the administration of morphine-like drugs.

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"The beginning of the physicians' secret: knowledge of the heart's movement and knowledge of the heart. There are vessels from it to every limb. As to this, when any physician, any surgeon, or any exorcist applies the hands or his fingers to the head, to the back of the head, to the hands, to the place of the stomach, to the arm or to the feet, then he examines the heart, because all his limbs possess its vessels, that is: the heart speaks out to the vessels of every limb."

Ebers Papyrus, 1552 B. C. (trans. Major, 1954)

"The emotion of joy and anger are injurious to the spirit . . . violent anger is hurtful to Yin, violent joy is hurtful to Yang. When rebellious emotions rise to Heaven, the pulse expires and leaves the body . . . One should feel the pulse at the place of the 'cubit' and at the place of the 'inch' and one should observe whether the pulse is superficial or whether it is deep, whether it is regular or uneven."

Nei Ching. The Great Treatise on the Interaction of Yin and Yang.

2698-2598 B. C.

"The chest in Living and perfect Animals being open'd and the Heart un-Capsulated, 'tis easy to observe a successive, or Reciprocal motion and Quiescency which Anatomist's call the contraction and dilatation of the Heart: which is more cleerly observable in frigid Animals which have not by farr so quick and smart Reciprocations as in froggs, snakes, vipers, toads and fishes and likewise in all languid and Dying Animals, for then the period of its motion are easyly discernible and the Perisystole or interjected Quiency is of a longer continuance."

Henry Power, *Circulatio Sanguinis* CAP. I, 1652.

Pulmonary Embolism

BILL FRANCOMBE, '58

INTRODUCTION

Pulmonary embolism is best considered a part of the process of venous thrombosis rather than a complication of this disease. Thinking in this manner serves to emphasize the importance of the prevention of venous thrombosis.

DEFINITION

Pulmonary embolism is the lodgement at some point in the pulmonary arterial tree of some foreign material, especially a blood clot which has broken off from a site of thrombosis in the deep veins of the pelvis or lower extremities.

SOURCE OF EMBOLI

1. Venous Thrombosis — This is by far the most common source. Ninety-five per cent of pulmonary emboli arise from areas of venous thrombosis in the deep veins of the pelvis or legs. This occurs most often from phlebothrombosis rather than from thrombophlebitis in which the thrombus is more adherent to the vessel wall due to the inflammatory reaction. Emboli from a superficial thrombophlebitis are said to be uncommon and rarely severe.
2. Thrombi developing in the right side of the heart after myocardial infarction or during auricular fibrillation.
3. Vegetations on the pulmonary and tricuspid valves and about septal defects. These emboli are small and multiple.
4. Fat and bone marrow embolism following crushing injuries and fractures.
5. Air embolism.
6. Oil embolism.
7. Amniotic fluid embolism.
8. Septic emboli—focal infection invading venous return.
9. Malignant emboli—malignant tumors invading veins and so metastasizing to lungs.

We shall limit ourselves to venous thrombosis in the deep veins of the pelvis and legs.

VENOUS THROMBOSIS

Two types of venous thrombosis are recognized:

1. Thrombophlebitis in which the inflammatory element predominates.
2. Phlebothrombosis in which the inflammatory element is absent or minimal.

In the first case the inflammation binds the thrombus to the vein wall decreasing the risk of embolization (5% embolize) whereas in the second case the thrombus floats free in the blood stream and so is more liable to break off (30% embolize). Also phlebothrombosis is more dangerous because it is so often unrecognized and so has more chance to embolize prior to commencement of active treatment. Thrombophlebitis and phlebothrombosis are essentially the same process except for the degree of inflammation present at any one time. Phlebothrombosis exists for a short time only before inflammation becomes apparent.

INCIDENCE

Thrombo-embolism occurs in about 1% of hospital patients. Pulmonary embolism

causes 2% of hospital deaths and is involved in 50% of important post-operative complications.

COURSE

Venous thrombosis may:

1. Organize and re-canalize so that venous circulation returns to normal. Adequate collaterals are important during this phase.
2. Completely block vein and spread to involve many veins leaving a chonic phlebitic limb.
3. Embolize.

PREDISPOSING CAUSES

General

Three factors enter into all predisposing conditions.

1. Local venous injury.
2. Venous stasis or slowing of venous return.
3. Increased clotting tendency.

Specific

1. Post-operative. Especially 5-15 days after abdominal and pelvic operations.
2. Long immobilization in bed for any reason, especially for fractures of the lower extremities.
3. Congestive heart failure — particularly if because of mitral stenosis or myocardial infarction.
4. Childbirth.
5. Age and general debility. In 50% of people over middle age thrombi are present in the deep veins of the legs. 93% of thrombo-embolic episodes occur in people over 40 years of age.
6. Infections. Patients with such as typhoid fever or pneumonia are prone to the development of venous thrombosis.
7. Dehydration.
8. Varicosities.
9. Malignancy. Carcinoma of the stomach and of body or tail of pancreas may be associated with widespread and migrating venous thrombosis.
10. Miscellaneous conditions include obesity, anemia, thrombo-angititis obliterans, leukemia and polycythemia vera.

SIGNS AND SYMPTOMS

Most cases of early phlebothrombosis are probably missed as the first sign in many patients is the occurance of a pulmonary embolus. There may be a slight ache in the limb and a feeling of fullness or warmth. Mild edema may be detected and Homan's sign (pain in the calf with dorsiflexion of the foot) may be present.

If thrombophlebitis is present, the pain in the limb may be slight or very severe, the limb tender to touch and a generalized edema with some cyanosis may be apparent. The patient with much inflammation at all will have an elevated temperature and pulse rate. Venograms can localize the site of thrombosis.

TREATMENT OF VENOUS THROMBOSIS

A. Prevention—this is the most important element in treatment.

1. Avoid unnecessary trauma during operative procedures, particularly in large abdominal and pelvic operations.
2. Avoid pressure or constricting binders on abdomen and legs during operation and period of bed rest.
3. Examine lower limbs of bed patients daily.
4. Active exercises—if possible patient should move legs actively while in bed.

5. Passive exercises and massage if active exercise impossible. Some believe massage may dislodge formed thrombi.
6. Deep breathing exercises carried out four times a day may stimulate venous return.
7. Change patients position every hour.
8. Correct pre-disposing causes such as dehydration and infection.
9. Early ambulation—this is very important and does not mean just sitting on the edge of the bed or on a chair but actual walking.
10. Anticoagulation—has been suggested and has been used as a prophylactic measure in patients prone to venous thrombosis, especially post-operative patients. However, anticoagulants are most commonly reserved for use when first signs of venous thrombosis appear.

B. ACTIVE TREATMENT

Once signs, however slight, of venous thrombosis are present, active treatment should be started aimed at preventing pulmonary embolism and avoiding chronic phlebitis.

1. Bed-rest—even though exercises and early ambulation are prominent features in prevention, bed-rest is advocated once signs appear.
2. Elevation of foot of bed—raising the legs 8 inches improves venous return and reduces edema.
3. Moist heat—warm moist packs applied to whole limb.
4. Anticoagulation seems to be excellent in the treatment of venous thrombosis. It helps avoid pulmonary emboli and extension of existing thrombi and seems to have a beneficial effect on the inflammatory process itself.
5. Early exercise and ambulation once signs and symptoms gone. An elastic stocking or bandage may be necessary in order to control edema when patient ambulatory.
6. Lumbar sympathetic block has been done to relieve arterial spasm which may be associated with venous thrombosis. However, moist heat seems to do just as well in this regard.
7. Parenteral trypsin and phenylbutazone are undergoing clinical trials. It is therefore too early to evaluate their usefulness.
8. Venous ligation has been considered as both a prophylactic measure and as a part of active treatment. It seems best used as the treatment of recurrent pulmonary emboli not responding to anticoagulation.

PULMONARY EMBOLISM

Signs and Symptoms

The clinical picture of pulmonary embolism is quite varied. It may present as a sudden chest pain with dyspnea, cyanosis and circulatory collapse or give no indication until the development of chronic cor pulmonale from repeated, silent emboli.

A large embolus causes acute cor pulmonale with precordial or subternal chest pain, cyanosis and shock. Death may come very rapidly. A systolic murmur and an increased pulmonic second sound may be heard at the second or third interspace to the left of the sternum. Jugular distension may be marked.

Moderate sized emboli may give transient chest pain and dyspnea followed by pleuritic pain, hemoptysis and signs of consolidation — others may present with pleuritic pain and hemoptysis only.

All pulmonary emboli do not cause pulmonary infarction. Massive emboli can cause death before infarction occurs. The lungs are well supplied with collaterals so infarction is not the ultimate outcome.

The patient suffering a pulmonary infarct will have leucocytosis, and an elevated pulse, temperature and sedimentation rate. Radiological evidence appears in 12-24 hours although the classical wedge-shaped area of consolidation is not commonly seen.

DIAGNOSIS

One must always consider pulmonary embolism as a possibility in a patient predisposed to venous thrombosis and in such a patient any chest pain or fever must be viewed with suspicion. Many clinicians consider the triad of sudden dyspnea, chest pain, aggravated by respiration and followed by hemoptysis as pathognomonic of pulmonary embolism. Acute cor pulmonale always suggests massive pulmonary embolism.

PATHOLOGICAL PHYSIOLOGY

The effect of pulmonary emboli is proportional to the circulatory upset and the size of the infarct, if present.

A. *Circulatory*

1. Obstruction to arterial flow. It is estimated that two-thirds of the pulmonary arterial flow must be blocked before the systemic circulation becomes impaired. Large emboli may be 30 cm. long and so project into the right ventricle impairing pulmonary valve function. Usually over 85% of the pulmonary arterial flow is blocked before death occurs from asphyxia.

2. Autonomic reflexes may produce pulmonary arterial spasm, bronchial spasm and coronary arterial spasm, leading to pulmonary hypertension and right heart failure with the picture of acute chest pain, dyspnea and shock.

Most emboli, however, do not grossly upset the circulation.

B. *Infarction*

If an arterial branch is blocked and the local collaterals are inadequate, a hemorrhagic infarct results. The infarct can become infected to form an abscess and a sero-sanguinous pleural effusion can result from the subsequent pleuritis.

DIFFERENTIAL DIAGNOSIS

1. *Myocardial Infarction.* This is most commonly confused with pulmonary embolus because of the picture of acute chest pain, dyspnea and shock. Fewer mistakes will be made if one first thinks of pulmonary embolism when the above symptoms occur in a predisposed patient. An electrocardiogram is important in the differential diagnosis. Serum transaminase levels, elevated in myocardial infarction but not in pulmonary infarction may prove to be important.

2. *Pleurisy*—suspect any pleural pain arising in a predisposed patient.

3. *Bronchopneumonia* — often frequent attacks of pneumonia have really been the result of a series of pulmonary emboli.

4. *Spontaneous Pneumothorax.*

5. *Neoplasm* — non-specific radiological findings in a person with a hemorrhagic effusion and hemoptysis may be thought to represent a pulmonary malignancy while actually these signs have followed a pulmonary infarction.

TREATMENT

A. *Prevention*

1. Prevention of venous thrombosis.
2. Prevention of emboli once veins involved i.e. active treatment of venous thrombosis.

B. *Active*

1. Immediate—if the embolus is large and possibly fatal, atropine 1 mgm., papaverine 30-60 mgm., and heparin 50 mgm. should be given quickly and intravenously. Oxygen by BLB mask should be given.

2. Subsequent—Atropine and papaverine may be repeated in three hours if necessary.

1) Bed-rest for 1 week at least until signs and symptoms gone.

2) Adequate anticoagulation continued for 1 week after bed-rest completed (minimum of 2 weeks).

3) Standard treatment for shock, congestive failure, infection and effusion if they develop.

4) Analgesia for pain, however morphine may depress already impaired oxygen uptake and carbon dioxide elimination.

5) Embolectomy has been performed for massive pulmonary embolism but the mortality rate for this operation is 90% as the patient is a poor risk to start with.

6) Venous Ligation — much confusion has existed regarding this aspect of therapy. Unilateral common femoral ligations were done if one limb was obviously affected with thrombophlebitis; however, the embolus may have arisen from the limb presenting no signs or symptoms. Bilateral common femoral ligations were then suggested but the feeling now seems to be if any venous ligation is to be done, it should be the inferior vena cava that is ligated and then only if adequate anticoagulation has failed to stop pulmonary emboli from recurring.

PROGNOSIS

Most patients survive the first pulmonary embolus but if no treatment is given 30% of these patients will suffer recurrent pulmonary emboli and half of these will die. If proper treatment is given, only 2% will suffer recurrences. If a patient survives one hour his chances improve considerably and if this is prolonged to 12 hours his chances are excellent.

CONCLUSION

Pulmonary embolism is a serious complication arising among medical, surgical and obstetrical patients. It is an integral part of thrombotic disease of the deep veins of the pelvis and lower limbs. Prevention of venous thrombosis is the most important factor in treatment. With treatment the incidence of pulmonary emboli can be greatly reduced.

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Medico-Legal Notes

DON BATES, '58

The lie-detector depends for its results upon the measurement of physiological changes in the tested person. Centuries before this became possible, however, many systems were designed to separate the true from the false. Trial by fire, by battle, and the use of mysterious potents were widely popular and equally useless methods used by the ancients. But the Hindus had perhaps one of the cleverest methods which depended, rather crudely perhaps, on superstition and psychology for its effectiveness. The suspect was told that a sacred ass would bray if its tail was grasped by a guilty person, but would remain silent if the party was innocent. The test was carried out in a room occupied only with the mule and suspect but within the hearing of the priests. Before the trial, and unknown to the contestant, lamp black was put on the ass's tail. The innocent person went in and grabbed the tail with impunity, thus blackening his hand. But the guilty party tried to avoid detection by leaving the room without touching the animal. Hence he emerged with incriminatingly clean hands!

Late in the afternoon of September 29th., 1935, four bundles containing human remains were found in a ravine near Moffat in Dumfrieshire, Scotland. Six months later, in March, on a Friday the 13th., Dr. Buck Ruxton was found guilty and sentenced to death for the murder of his wife and nursemaid. The investigation which led to this conclusion represents one of the most careful studies ever performed by medico-legal experts.

The celebrated Ruxton case is perhaps most notable for the use of photographs in reconstructing portraits from the skulls found and thereby identifying the victims. Though skulls and portraits were compared with a view to identification as early as 1918 it was not until this case in 1935 that such a painstaking comparison by super-imposition, matching scale, and scientific regard for similar position was made. Though the results did not prove conclusively (and possibly never can in normal people) that the skulls were those of the women in the photographs, they did not disprove the possibility and indeed were highly suggestive.

Skull studies as a means of identification are a relatively old innovation but prior to 1900 were more frequently applied to the identification of the remains of famous people. In some cases, the studies of skulls of known identity were used to prove or disprove the authenticity of portraits and busts purported to represent celebrities. This is true of Dante (1265-1321) whose skull matched a reputed death-mask of him. But the skull of Raphael (1483-1520) came back from the grave after 300 years to discredit two portraits and a mask claimed to be his likeness. The bones of Bach some hundred years after his death allowed Seffner to construct his bust which excelled all the portraits in its life-like character.

In other instances authentic photographs have been compared with skulls claimed to be of the same identity. The supposed skull of Lord Darnley, ill-fated husband of Mary, Queen of Scots, was matched with five genuine pictures, but the results were equivocal. The famed "Wilkinson Head" proved to be that of Cromwell (1599-1658) after one of the most extensive examinations ever undertaken.

Many advocates of this method of checking the validity of portraits, paintings, engravings and the like have been thwarted to date (i.e. 1937) in using Shakespeare's skull for this purpose because the inscription on his tombstone bears the instructions "forbeare to dig the dust enclosed heare," and curse on him "that moves my bones." Perhaps he was being mindful of "poor Yorick".

News and Views

TED SILLER, '59

Urology Award

The American Urological Association offers an annual award of $1000 (first prize $500, second prize $300, third prize $200) for essays on the result of some clinical or laboratory research in urology. Competition shall be limited to urologists who have graduated not more than ten years previously, and to hospital interns and residents doing research in urology. The prize essay will appear on the program of the forthcoming meeting of the American Urological Association, to be held at the Roosevelt Hotel, New Orleans, La., April 28 to May 1, 1958.

For full particulars write the executive secretary, Wm. P. Didusch, 1120 North Charles St., Baltimore, Md. Essays must be in his hands before December 1, 1957.

Doctors on Strike

Several important Swedish towns have been exposed to the conflict between the Swedish medical profession and the municipal authorities concerning remuneration. At one hospital there was withdrawal of many physicians from their appointments after due notice. Consultations and treatment were arranged outside the hospital and a private surgical clinic has been treating several hundred patients daily. A skeleton staff has remained at the hospital to prevent victimisation of the public. This strike at last word was spreading to larger Swedish centres.

Book Reviews

SURGERY PRINCIPLES AND PRACTICE

J. Garrott Allen, University of Chicago; Henry N. Harkins, University of Washington; Carl A. Moyer, Washington University, St. Louis, and Jonathan E. Rhoads, University of Pennsylvania. 1495 pp. illust. J. B. Lippincott Company, Montreal, 1957. $16.00.

This is an entirely new textbook, written to provide the medical student with a background knowledge of anatomy, pathology, physiology and biochemistry in order to enable him to develop acumen in all phases of surgery. In other words, the authors wanted to present the principles of surgery, which I believe they have accomplished quite adequately.

The text is well planned and easily read. It contains many drawings, radiographs, charts and photographs which are well placed and well done. At the beginning of each chapter the authors present an outline of the chapter which is very useful to the student as it enables him to read with the discretion necessary due to his limited time.

Of special interest are chapters entitled surgical philosophy, surgical infections, fluid and electrolytes, and non-operative surgical care.

The only fault of the text is one which is shared by almost all present-day textbooks in that it is printed on expensive, hard-on-the-eyes, glossy paper. However, this book serves the student's need admirably and will likely be widely read.

Alumni and Faculty News

National and international recognition of the contributions and abilities of our faculty and alumni is a source of pride, not only to themselves, but to this university community as well.

Doctors Murray and Robinow were joint recipients earlier this year of the Harrison Prize, awarded by the Royal Society of Canada. They received the prize in recognition of contributions, of a basic, non-medical nature, on the structure of bacteria. Dr. Robinow and Dr. Murray were the first recipients of this prize which will be awarded every three years.

Although we were sorry to lose him, our best wishes go with Dr. W. R. Begg who was formerly the National Cancer Institute Professor of Medical Research and Lecturer in Biochemistry at Western. Dr. Begg was chosen as director of the new $400,000 Saskatchewan Cancer and Medical Research Institute at Saskatoon. He will also assume the post of Professor of Cancer Research at the University of Saskatchewan.

Dr. Barr attended a Symposium on Nuclear Sex at London, Eng., Sept. 6th and 7th, where he gave a paper "Anomalies of Sex Development". This symposium, on the field in which Dr. Barr has made such basic and notable contributions, was widely attended.

London was well represented at the Second International Congress for Psychiatry, held in Zurich early in September. The conference, whose theme was "the present status of our knowledge about the group of schizophrenias", was attended by Doctors Hobbs, Goddard, McCausland and Prince (and by a certain dissociated member of Meds '58). Dr. Ray Prince (Meds '50) journeyed from Zurich to Africa to take up his new position as psychiatrist to the Abeokuta Psychiatric Hospital in West Nigeria.

We welcome to our school Dr. Robert Arthur Hugh Kinch, the new Professor of Obstetrics. Dr. Kinch, a popular and able teacher, came to us this summer when Dr. W. P. Tew retired to private practice and a well-deserved rest. Dr. Kinch graduated in 1943 from the Middlesex Hospital Medical School of the University of London. He served in the Royal Navy during the war and returned to do postgraduate work at Middlesex and Queen Charlotte Hospitals in London, after which he received his M.R.C.O.G. He came to Canada in 1948 and served on the staffs of the Toronto Western and Grace Hospitals and the University of Toronto. He was made a Fellow of the Royal College of Surgeons (Canada) in 1952.

A popular London physician and graduate of the UWO Medical School (Meds '44) Dr. William A. Banghart met a tragic death Sunday, August 4, 1957. Dr. Banghart and a companion were drowned off Port Stanley while on a fishing trip on Lake Erie. Dr. Banghart, whose interest was the field of allergy, will certainly be missed in London's medical circles. We would extend our sympathies to his family and friends.

Hugh W. Edgar
Meds '58

Recent Acquisitions in the Library

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